

Heat and Low-Density Lipoprotein Transfer in Healthy Aorta using Two-Way Fluid-Structure Interaction Method

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Abstract- This study aims to reveal the mechanism of heat and low-density lipoprotein (LDL) transfer in healthy aortas leveraging a fluid-structure interaction model. Two healthy aortic geometries are reconstructed. The pulsation and non-Newtonian viscosity of blood flow are considered. The aortic wall is assumed to be hyperelastic. Results show that there is a strong positive correlation (>0.596) between the LDL and wall temperature. The long relative residual time region also coincides with the high LDL, which negatively correlates with wall shear stress and topological shear variation index. This study would inform novel strategies to measure LDL concentration and regulate its accumulation.

Key words: Computational hemodynamics, Temperature distribution, Atherosclerosis

I. INTRODUCTION

The cardiovascular circulatory system plays a crucial role in maintaining human life and health by transporting nutrients and metabolic wastes [1]. The biggest blood vessel of the cardiovascular circulatory system is the aorta, which has a high risk of atherosclerosis. The penetration and accumulation of low-density lipoprotein (LDL) into the vessel wall are the main contributing factors to atherosclerosis [2]. However, the mechanism of atherosclerosis and LDL transfer in the aorta remains unclear.

The high aortic blood flow energy loss region localizes with aortic atherosclerosis-prone areas [3, 4]. The consumed energy of the blood flow is converted into heat and may alter the distribution of temperature, which has been reported in our recent study [5]. Therefore, there is supposed to be a relationship between the aortic temperature distribution and atherosclerosis, which mainly results from the penetration and accumulation of LDL into the aortic wall. The present study focuses on the transfer process of heat and LDL.

This study aims to investigate the heat and LDL transfer in healthy aorta. Firstly, we reconstructed two healthy aortic geometry models based on computed tomography angiography (CTA) images, and patient-specific boundary conditions were acquired according to routine clinical measurements. Then, the control equations of heat and LDL transfer were numerically solved using the two-way FSI method. Finally, the crucial hemodynamic

parameters were reported and we also demonstrated the relationship between the LDL and hemodynamics, especially the wall temperature. This study provided a novel insight into further understanding of the human aorta and found an alternative method for measuring LDL using temperature.

II. METHODOLOGY

Geometry construction and mesh generation

CTA images of two healthy aortas were offered by the Zhongshan Hospital Affiliated with Fudan University (China) with the approval of the Ethics Committee. The three-dimensional aortic geometry (blood flow domain) was reconstructed based on clinical CTA images using commercial software Mimics 19.0 (Materialise, Leuven, Belgium). The aortic vessel wall was also created by assigning a thickness of 2 mm to the inner surface of the aortic geometry. Figure 1 shows the CTA images and three-dimensional aortic geometry.

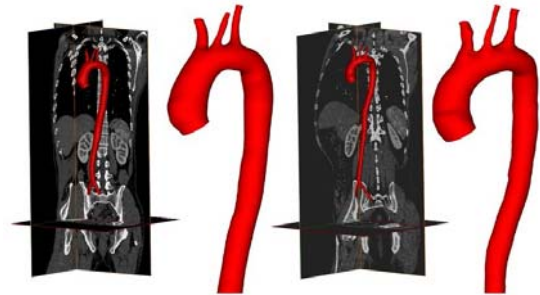


Fig. 1. CTA images and three-dimensional aortic geometry.

Both the aortic geometry and vessel wall were divided into unstructured tetrahedral meshes using ANSYS-ICEM (ANSYS Inc., Canonsburg, PA, USA). For the blood flow domain, the maximal mesh size was 4×10^{-4} m. Twenty prism boundary layers with a growth rate of 1.3 were designed to capture the hemodynamics near the aortic wall. The height of the first boundary layer was set to 8×10^{-6} m to accurately predict the LDL concentration [6]. The present mesh strategy was the strictest relative to our previous studies, where the mesh sensitivity tests were satisfied.

B. Interaction of blood flow and aortic wall

Blood was simplified to be an incompressible fluid with a density of 1080 kg/m^3 . The non-

Newtonian Quemada model was adopted to calculate the viscosity of the blood flow. A clinical blood flow waveform with a three-dimensional parabolic profile was set as the inlet boundary condition [7]. Three-element Windkessel model was used to predict the outlet pressure waveform. The shear stress transport model was chosen to capture the turbulent flow and a turbulence intensity of 1.5% was specified at the inlet of the ascending aorta. A bilateral FSI computational model was developed to consider the interaction between the blood flow and the aortic wall based on our previous studies [3-5, 8, 9]. The Yeoh 2nd Order hyperelastic model was adopted to describe the behavior of the aortic wall and the parameters were from a previous work [10]. The FSI model was implemented on ANSYS Workbench (ANSYS Inc., Canonsburg, PA, USA), where the Transient Structural (aortic wall) was coupled with CFX (blood flow). More details of the FSI model can be found in our latest studies [5, 8].

C. Heat and LDL transfer

The specific heat capacity of the blood (c_p) was 4000 J/Kg/K and the thermal conductivity (k_T) was 0.6 W/m/K [11]. The temperature of the aortic inlet was set to 310 K. A uniform heat influx of 10 W/m² was applied at the aortic wall to model the heat generated by surrounding tissues [12, 13].

The convection-diffusion transport equation was applied to describe the LDL transfer:

$$\frac{\partial C}{\partial t} + u_b \nabla C = D_{LDL} \nabla^2 C$$

where C is the LDL concentration and D_{LDL} is the diffusion coefficient. The LDL concentration at the aortic inlet was 1.2 kg/m³ and zero-flux condition for LDL was applied at the aortic outlets. The LDL transfer between the blood flow and aortic wall was considered:

$$C_w V_w = D_{LDL} \left. \frac{\partial c}{\partial n} \right|_w$$

where C_w is the LDL concentration on the aortic wall, V_w is the water filtration velocity, and $\left. \frac{\partial c}{\partial n} \right|_w$ is the LDL concentration gradient normal to the aortic wall.

Following the practice of a previous valuable study [6], a steady simulation was first carried out to get the initial LDL distribution. Then, the unsteady FSI simulation with a timestep of 5 ms was carried out. The required computational cardiac cycle for the LDL concentration to reach a periodic state was 11 and 7 for the two patients, respectively.

D. Hemodynamic Parameters

To reveal the mechanism of aortic heat and LDL transfer, the relationship between the LDL and the following hemodynamic parameters was analyzed. The LDL and temperature on the aortic wall were time-averaged (TALDL and TAT) during a cardiac cycle. WSS-related indices were incorporated, such as time-averaged wall shear stress (TAWSS),

oscillatory shear index (OSI), relative residence time (RRT), and topological shear variation index (TSVI) [14, 15].

III. RESULTS AND DISCUSSION

A. TALDL vs. TAWSS

Figure 2 shows the relationship between the TAWSS and TALDL on the aortic wall, where the values are normalized based on maximum and minimum. A relatively strong negative correlation (0.690 and 0.648) is observed. The aortic wall with high TALDL is exposed to low TAWSS. The high TAWSS region is accompanied by a relatively low TALDL concentration. Most points are concentrated in the high TALDL and low TAWSS regions.

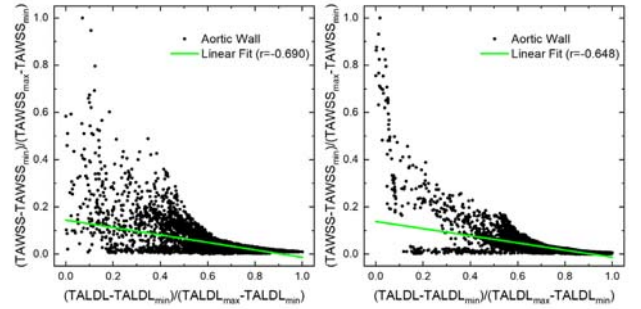


Fig. 2. The relationship between the TAWSS and TALDL on the aortic wall.

B. TAT vs. TALDL and TAWSS

The relationship between the TAT and TALDL on the aortic wall is analyzed in Fig. 3(a). The values of the TAT and TALDL are normalized based on the corresponding maximum and minimum. A relatively strong positive correlation (0.729 and 0.596) is observed. The low and high values of TAT correspond well to the concentration of TALDL. However, the medium values of the TAT and TALDL show slight deviation.

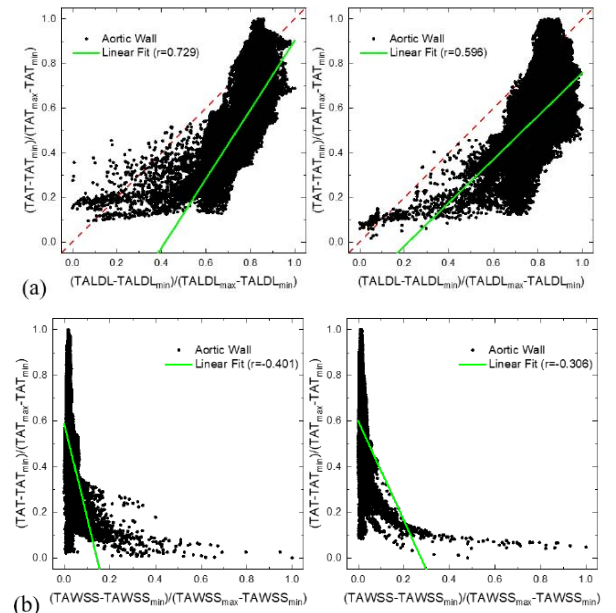


Fig. 3. (a) The relationship between the TAT and TALDL. (b) The relationship between the TAT and TAWSS.

Figure 3(b) further explores the relationship between the normalized TAT and TAWSS on the aortic wall. There is a moderate negative correlation (0.401 and

0.306), which is relatively lower than the correlation coefficients between TALDL and TAWSS, TALDL and TAT. The aortic wall with high temperature is exposed to low TAWSS. The high TAWSS region overlaps with the low-temperature area. Most points are concentrated in the low TAWSS region.

C. TALDL vs. RRT and TSVI

The relationship between the RRT and TALDL on the aortic wall is revealed in Fig. 4(a). A relatively weak positive correlation (0.308 and 0.324) is observed. The aortic wall with long RRT coincides with high TALDL concentration. Low TALDL region shows short RRT. Most points of the aortic wall are under the high TALDL and low RRT environment.

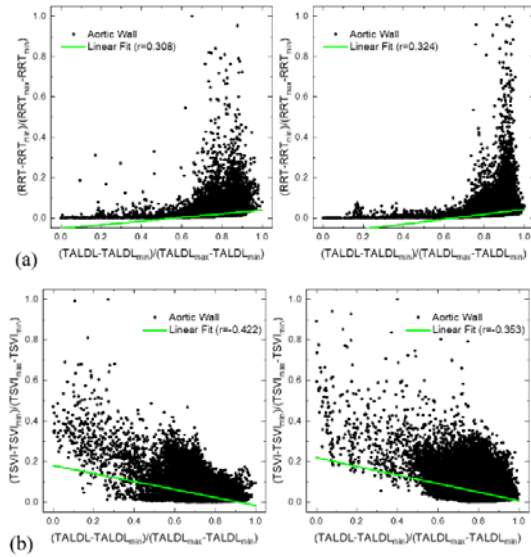


Fig. 4. (a) The relationship between the RRT and TALDL. (b) The relationship between the TSVI and TALDL.

Figure 4(b) investigates the relationship between the TSVI and TALDL on the aortic wall. There is a weak negative correlation (0.422 and 0.353). The aortic wall with relatively high TALDL concentration is consistent with low TSVI. The high TSVI region is accompanied by low TALDL. Most points are concentrated in the high TALDL and low TSVI regions.

IV. CONCLUSIONS

This study investigates the heat and LDL transfer in healthy human aorta based on a two-way FSI method. The major finding is that there is a strong positive correlation between the LDL concentration and wall temperature. Besides, the aortic wall with long RRT is also exposed to high LDL and both WSS and TSVI negatively correlate with LDL. In conclusion, the mechanism of heat and LDL transfer in the human healthy aorta is demonstrated, providing a basis for developing novel measurement methods to acquire LDL concentration and avoid its accumulation.

REFERENCES

[1] A. Stefanovska. Physics of the human cardiovascular system. *Contemporary Physics*, 40(1): 31- 55, 1999.
 [2] L.B. Nielsen. Transfer of low density lipoprotein into the arterial wall and risk of atherosclerosis. *Atherosclerosis*, 123(1-2): 1-15, 1996.

[3] Y. Qiao, K. Luo, J. Fan. Component quantification of aortic blood flow energy loss using computational fluid-structure interaction hemodynamics. *Comput. Methods Programs Biomed.*, 221: 106826, 2022.

[4] Y. Qiao, L. Mao, Y. Ding, T. Zhu, K. Luo, J. Fan. Fluid-structure interaction: Insights into biomechanical implications of endograft after thoracic endovascular aortic repair. *Comput. Biol. Med.*, 138: 104882, 2021.

[5] Y. Qiao, K. Luo, J. Fan. Heat transfer mechanism in idealized healthy and diseased aortas using fluid-structure interaction method. *Biomechanics and Modeling in Mechanobiology*, 22: 1953-1964, 2023.

[6] G. De Nisco, P. Zhang, K. Calò, X. Liu, R. Ponzini, C. Bignardi, G. Rizzo, X. Deng, D. Gallo, U. Morbiducci. What is needed to make low-density lipoprotein transport in human aorta computational models suitable to explore links to atherosclerosis? Impact of initial and inflow boundary conditions. *J. Biomech.*, 68: 33-42, 2018.

[7] J. Alastruey, N. Xiao, H. Fok, T. Schaeffter, C.A. Figueroa. On the impact of modelling assumptions in multi-scale, subject-specific models of aortic haemodynamics. *J R Soc Interface*, 13(119): 20160073, 2016.

[8] Y. Qiao, J. Fan, K. Luo. Mechanism of blood flow energy loss in real healthy aorta using computational fluid-structure interaction framework. *International Journal of Engineering Science*, 192: 103939, 2023.

[9] Y. Qiao, J. Luan, L. Mao, J. Fan, T. Zhu, K. Luo. Biomechanical mechanism of distal stent-graft-induced new entry deterioration after thoracic endovascular aortic repair. *Phys. Fluids*, 34(10): 101902, 2022.

[10] M. Raghavan, D.A. Vorp. Toward a biomechanical tool to evaluate rupture potential of abdominal aortic aneurysm: identification of a finite strain constitutive model and evaluation of its applicability. *J. Biomech.*, 33(4): 475-482, 2000.

[11] J. Crezee, J. Lagendijk. Temperature uniformity during hyperthermia: the impact of large vessels. *Phys. Med. Biol.*, 37(6): 1321, 1992.

[12] H. Kang, S.A. Jasim, S.N. Sedeh, M. Hekmatifar, D. Toghraie, W. Suksatan, S. Raheem, O. Viktorovna Dudnik. Heat transfer and hemodynamic analysis of systolic and diastolic hypertension on abdominal aortic thrombosis. *Case Studies in Thermal Engineering*, 30: 101738, 2022.

[13] Y.-M. Li, S.N. Sedeh, D. Toghraie, A.a. Alizadeh. Computational hemodynamics and thermal analysis of laminar blood flow for different types of hypertension. *Mathematics and Computers in Simulation*, 188: 330-341, 2021.

[14] U. Morbiducci, V. Mazzi, M. Domanin, G. De Nisco, C. Vergara, D.A. Steinman, D. Gallo. Wall Shear Stress Topological Skeleton Independently Predicts Long-Term Restenosis After Carotid Bifurcation Endarterectomy. *Ann. Biomed. Eng.*, 48(12): 2936-2949, 2020.

[15] V. Mazzi, D. Gallo, K. Calò, M. Najafi, M.O. Khan, G. De Nisco, D.A. Steinman, U. Morbiducci. A Eulerian method to analyze wall shear stress fixed points and manifolds in cardiovascular flows. *Biomechanics and Modeling in Mechanobiology*, 19(5): 1403-1423, 2020.